Pontiac Fever Outbreak Associated with a Cooling Tower

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Abstract: In late April 1984, an outbreak of Pontiac fever was investigated in an office buiding in lower Manhattan (New York City). The outbreak was characterized by a high attack rate (78 per cent overall); the predominant symptoms were myalgias, chills, fatigue, fever, and headache. There was a clustering of cases in an office that was air cooled by a dedicated cooling tower separate from the remainder of the building. A high concentration of live *L. Pneumophila* cells in the cooling tower was quantified. Airborne spread via settle plates placed along the air intake system and within the office was demonstrated. *Legionella pneumophila* serogroup 1 antigen was found in the urine of two cases, and identical monoclonal antibody reactivity patterns of isolates from all sources was observed. Difficulty was experienced in eliminating the organism from the tower. (*Am J Public Health* 1987; 77:568–572.)

Introduction

Legionellosis or illness caused by *Legionella* species demonstrates two clinicoepidemiologic patterns: Legionnaires' Disease, a multisystem illness characterized by pneumonia with a case-fatality rate of 6 per cent or more[1,2]; and Pontiac fever, a self-limited illness characterized by fever, headache, myalgia, and fatique.[3] Five point-source outbreaks of Pontiac fever have been reported: in Pontiac, Michigan in 1968 from a defective air conditioning system[3]; in James River, Virginia in 1973 resulting from compressed air used to clean a steam turbine engine[4]; in Vermont in 1981 from a whirlpool spa[5]; in Windsor, Canada in 1981 from a water-based coolant in an engine assembly plant[6]; and in Rochester, Michigan in 1982 from a whirlpool spa.[7] In contrast to the other four outbreaks, a non-pneumophila *Legionella* species, *L. feeleii* was implicated as the etiologic agent in the Canadian outbreak. In each of these outbreaks, Pontiac fever has presented as a non-pneumonic, influenza-like illness with a short incubation period (approximately 36 hours), a high attack rate (100 per cent in one outbreak), transmission by aerosol from a heated water source, and a lack of secondary spread.

Background

In the spring of 1984, the New York City Department of Health investigated an outbreak of Pontiac Fever caused by *L. pneumophila* serogroup 1 in a business office in lower Manhattan. The office, a branch of a bond processing company, occupies four floors in a 20-story building. The lower two floors are below ground level and include a large vault where bonds are stored. The remaining two floors are the ground and mezzanine. A 55-ton capacity cooling tower installed in November 1983 supplies only these four floors; thus air movement, heating, and cooling are separate from the remainder of the building. The make-up water for the cooling tower is the potable city water. The cooling tower had been in operation continuously since it also supplied hot water to heat the offices in winter. Before and during the outbreak period, the cooling tower functioned to cool the offices.

Fresh air for the ground floor and mezzanine is introduced by a fan in the air conditioner located on the mezzanine level. The fresh-air intake is located 4 meters from the cooling tower exhaust louvers; both are located on the mezzanine level. The air conditioning fan draws an equal mixture of fresh air and recirculated office air and distributes it to the two upper floors through a series of ducts. Although another system for drawing fresh air to the two lower floors exists, it was not in operation two weeks prior to and during the outbreak. Air intake for the lower two floors including the vault area was through the stairway joining these areas with the upper floors.

The company staff is composed of approximately 80 people involved in clerical work and eight security guards. The work schedule is weekdays from 8:30 AM to 4:30 PM and, if necessary, Saturdays.

On Monday morning, April 30, 1984, the company noted that the majority of 31 employees who had worked Saturday, April 28 were ill with fever and headache. Employees who worked Saturday were seen by the company's employee health service nurse who reported the outbreak to the New York City Department of Health. In response to the outbreak, the company closed the office to employees at 4 PM on April 30 and reopened on May 14. However, investigators continued to visit the office during this period. Investigators and visitors to the office on May 1 and May 2 wore no masks or protective equipment.

Methods

Department of Health employees administered a written questionnaire to all office employees and to people who visited the office from April 28 to May 5. The questionnaire was used to obtain information about demographics, presence and duration of symptoms and signs of illness, treatments used, history of food and water consumption, usual desk location, time spent on each of the four floors, cigarette smoking, and history of illness among household members.

A case of Pontiac fever was defined clinically as either chills or fever *and* either muscle aches or joint pains in a person who had worked or visited the building between April 23 and May 4. Although there is no uniform clinical case definition for Pontiac fever, this definition is similar to that used in previous outbreaks. Serologic confirmation defined as a four-fold rise in antibody to a titer of at least 1:128 was not required for inclusion as a case.

Paired serum specimens (separated by a mean of 40 days, range 14–64 days) were collected from office employees, visitors to the office, and from individuals who were in the building but not in the office on April 28. Serum specimens were tested at the Centers for Disease Control (CDC) using the indirect fluorescent antibody (IFA) test for *L. pneumo-*

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phila (Philadelphia strain), *L. pneumophila* (environmental isolate from the cooling tower at the outbreak site), *L. gormanii* (LS-13 strain), and *L. feeleii* (WO-44c strain).[8]

Urine and serum specimens were tested for soluble antigens of *Legionella pneumophila* by enzyme-linked immunosorbent assay (ELISA) tests with polyclonal and monoclonal antibodies to *L. pneumophila* serogroup 1. Immulon 2* microtiter plates (Dynatech Laboratories, Alexandria, VA) were activated with polyclonal antibodies to *L. pneumophila* serogroup 1 (Knoxville 1 strain). Urine and serum specimens were incubated in the activated wells and tested for immobilized antigen in an indirect ELISA system with monoclonal antibodies to *L. pneuomophila* serogroup 1 (monoclonal antibodies 1, 2, and 4) by procedures described previously.[9]

Water samples were collected from the cooling tower supplying the office and from drinking fountains, as well as water condensates found along the air handling system and in heat pump condensation trays. All were cultured and examined by the direct fluorescent antibody (DFA) test for *Legionella* species.[10,11] Also, the cooling tower water was examined microscopically for protozoa since some legionellae may grow intracellularly in protozoa.[12] The autoimmune radiographic procedure was used to quantitate the numbers of *L. pneumophila* serogroup 1 on bacteriologic plates with heavy growth of legionellae.[13]

Monoclonal antibody reactivity patterns of epidemic-associated isolates of *L. pneumophila* serogroup 1 were determined by IFA testing with 9 monoclonal antibodies with unique specificity for *L. pneumophila* serogroup 1 antigens by a previously described method.[14,15]

On May 1, May 2, and again on May 10, while the cooling tower and the air handling system were in operation, settle plates using buffered charcoal yeast extract agar with alpha-ketoglutarate, glycine, polymixin B, anisomycin, and vancomycin supplements[10] were placed open for 30 minutes on desk tops located directly beneath air vents and in the air conditioning unit that cooled the fresh and recirculated air mixture.

On April 30, the filters on the main air conditioning unit on the mezzanine level were removed and sent to CDC for culture and DFA examination for *Legionella*. Filters were changed monthly by the maintenance company. The main air conditioning filters from the mezzanine unit which had been in use for the month of June were removed and sent to CDC along with a new, unused filter as a control for culture and DFA testing.

Statistical Analysis

Confidence intervals (95%) for the odds ratio were determined using the Miettinen exact procedure programmed by Rothman and Boice.[16] The difference in mean duration of time spent in the office and in geometric mean titers was determined by the t-test procedure of the Statistical Analysis System.[17]

Results

A total of 63 (83 per cent) of 76 office employees and 23 (68 per cent) of 34 visitors had illness that met the case definition. The symptoms of the cases are presented in Table 1. The median duration of illness was four days (range 1–13 days). The median age of patients was 29 years.

TABLE 1—Symptoms of Cases (n = 86), Pontiac Fever Outbreak, New York City, 1984

Symptom	Number	Per Cent
Myalgias	83	97
Chills	80	93
Fatigue	78	91
Fever	76	88
Headache	75	87
Backache	64	74
Arthralgias	50	58
Nausea	43	50
Chest Pain	40	47
Cough	38	44
Abdominal Cramps	31	36
Urinary Frequency	24	28
Diarrhea	21	24
Urinary Urgency	15	17
Vomiting	12	14

The distribution of the onset of cases is presented in Figure 1. Since many employees experienced exposure to office air over several hours on both April 28 and April 30, a precise incubation period could not be calculated. However, a maximum incubation period was determined using as the moment of exposure 8 AM of the earliest day worked beginning with April 28. Two cases whose onset dates preceded their proposed exposure periods were omitted. By this calculation, the median incubation period was 35 hours.

Attack rates varied by day of exposure (Table 2) and by duration of exposure. Saturday workers spent approximately seven hours in the office. Monday workers spent approximately five hours there, and visitors on Monday, April 30 through Wednesday, May 2 spent generally less time in the office. Mean duration of time spent in the office was higher for cases than for employees or visitors who did not meet the case definition. Employees or visitors who spent at least three hours in the office over the outbreak period were significantly more likely to qualify as cases (odds ratio = 6.25, 95 per cent CI = 2.07, 18.37). The day of exposure and duration of exposure variables were too closely associated to allow one to estimate which of these was a better predictor of illness.

Factors *not* affecting attack rates included age, gender, racial-ethnic group, drinking water from office fountains, history of smoking, and usual desk location.

Cases were no more likely than well people to report subsequent illness in household contacts.

Paired sera were collected on 71 people (55 office employees, seven visitors to the office, and nine individuals who had been in the building but not in the office on April 28). Twenty-two employees and one visitor showed a four-fold rise in titer to a level of at least 1:128 by IFA to the outbreak isolate, *L. pneumophila* serogroup 1 Manhattan-5 compared to none of nine individuals without office exposure (odds ratio = infinity, 95% CI = 1.35, ∞). Those meeting the case definition were more likely to seroconvert (22/54 vs 1/8), (odds ratio = 4.81, 95% CI = 0.66, 113.89). Cases had a higher geometric mean titer than non-cases (1:84.0 vs 1:41.5, p = 0.03). Employees or visitors who spent at least three hours in the office were more likely to seroconvert (23/56 vs. 0/6, odds ratio = ∞, 95% CI = 0.97, ∞). No difference in seroconversion rates was found between those who worked Saturday, April 28 (9/24, 38 per cent) and those who worked Monday, April 30 but not Saturday, April 28 (14/33, 42 per cent).

Urine samples on 2 of 16 cases gave positive reactions

*Use of trade names is for identification only and does not imply endorsement by the New York City Department of Health, the Public Health Service, or the US Department of Health and Human Services.

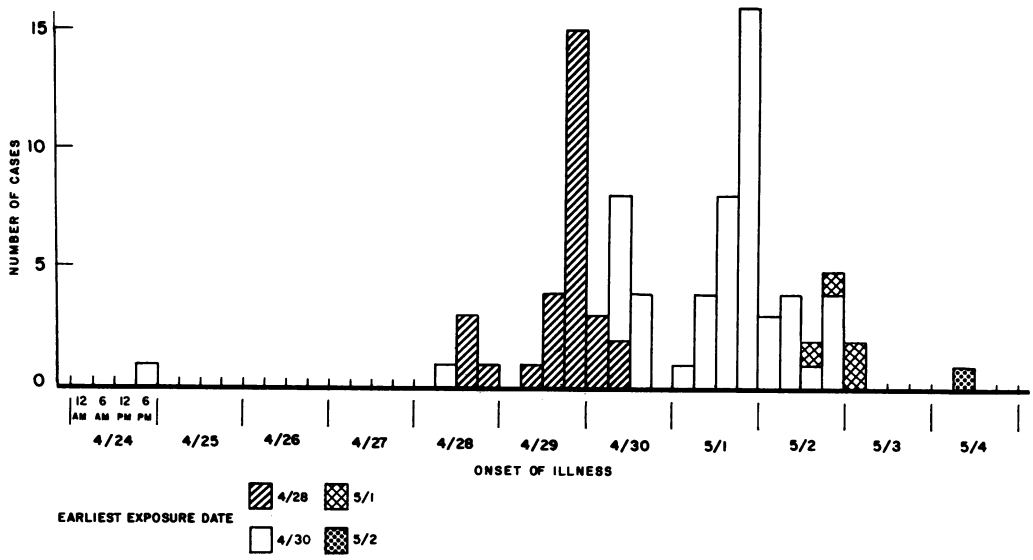

FIGURE 1—Epidemic Curve of Pontiac Fever Cases, New York City, April–May 1984

with monoclonal 2 antibody by indirect ELISA. All serum samples were negative in this test. Paired sera were available on one of these positive employees and showed a four-fold rise by IFA to 1:64 to the outbreak strain, but did not meet the 1:128 level required to be considered a seroconversion.

Environmental Results

Samples of water from the cooling tower supplying the office taken on Monday, April 30 and Tuesday, May 1 were positive initially for the outbreak strain of *L. pneumophila* serogroup 1 in concentrations of approximately 3×10^5 organisms/mL by DFA and by culture. On the evening of May 1, 1984 the cooling tower was treated with detergent, maintained at a level of 10ppm of free residual chlorine for 12 hours, drained, and brought to a level of 2ppm of free residual chlorine. Despite this treatment, cooling tower water samples remained positive on May 2, albeit at a level of 15 serogroup 1 organisms/mL by culture and approximately 3.6×10^4 organism/mL by DFA. On May 3, approximately 600cc of a butyl tin quaternary ammonium mixture and 2.2 kg of sodium chromate were added to the cooling tower by a commercial chemical company to further decontaminate the tower. The company estimated that the concentrations of tributyl tin and quaternary ammonia in the tower were 15 ppm and 45 ppm, respectively. Water samples taken from the cooling tower later the same day were positive for serogroup 1 at a level of 110 organism/mL by culture and 300 to 650 organisms/mL by DFA. Cooling tower water samples taken on May 4 were negative for *L. pneumophila* by culture, but DFA results showed approximately 1×10^3 organisms per mL. The cooling tower was hyperchlorinated to a level of 10 ppm on May 7 and was maintained at this level of 10 ppm for approximately 60 hours from May 11 to May 14. It was then kept at 2–10 ppm of chlorine throughout the summer. The sodium chromate level was maintained at 300–400 ppm. The NYC Department of Health sampled water from the cooling tower at two- to four-week intervals throughout the summer, and no legionellae were found by DFA testing or culture.

The air sampling plates placed on May 1 were positive for *L. pneumophila* serogroup 1 at all sites. Colony counts ranged from 20–80 per plate. Repeat testing at the same sites on May 2 and May 10 were negative.

The main air conditioning filters from the mezzanine level unit removed on April 30 were positive for *L. pneumophila serogroup 1* by DFA testing but not by culture. The filters from June 27, used and unused, were negative.

L. pneumophila serogroup 1 was also isolated from a drinking fountain, a hot water faucet, and a heat pump pan. The concentration of *L. pneumophila* in these was <1 CFU/mL. The immune autoradiographic procedure confirmed that all serogroup 1 *L. pneumophila* on the primary isolation plates tested contained the monoclonal 2 antigen.

All epidemic-associated *L. pneumophila* isolates gave identical 1, 2, 4, 5 monoclonal antibody reactivity patterns in IFA testing.

No protozoa were seen or cultured from cooling tower water samples.

Discussion

Pontiac fever was transmitted here by exposure to the office environment. Culture and DFA testing results demonstrated that the cooling tower supported the growth of *L. pneumophila*. A single source of *L. pneumophila* in the environmental waters sampled is suggested by the finding that all *L. pneumophila* cultures from the water samples were

TABLE 2—Attack Rates by Day of First Exposure

	Saturday April 28			Monday April 30			Tuesday May 1			Wednesday May 2		
Duration	<3 hrs	≥3 hrs	T	<3 hrs	≥3 hrs	T	<3 hrs	≥3 hrs	T	<3 hrs	≥3 hrs	T
Number Exposed	1	30	31	14	52	66	5	3	8	0	2	2
Attack Rate	0	97	94	57	83	77	40	67	50	—	50	50

the same monoclonal subgroup, reactive with monoclonal antibodies 1, 2, 4, and 5. We believe that organisms were distributed to the street air in the cooling tower exhaust vapor and taken into the office air via the fresh air intake point four meters away. Wind patterns along the street are erratic, but one Health Department investigator reported that on May 3 he observed mist from the cooling tower exhaust form and travel down-wind to the fresh air intake point where it was drawn into the building. Finding organisms on settle plates placed in the path of the air intake and on the air conditioning filter processing this air strongly supports this hypothesis. Airborne spread of the disease is further supported by our finding organisms on settle plates placed below air duct vents on the first floor.

Since the potable water also contained *L. pneumophila,* albeit in much lower concentrations than the cooling tower water, the potable water could have been responsible for seeding the cooling tower with the outbreak strain. We believe that legionellae in potable water multiplied in the cooling tower. Although no protozoa were observed in the cooling tower water, they may have been present prior to our sampling and provided a host for multiplication.[18]

We were unable to demonstrate that any area of the office was at increased risk for illness. Since the principal fresh air intake was the same for all floors in the office and since air circulated throughout the floors, organisms were likely to have been distributed to all levels. We attempted to determine a minimum effective exposure period but were hampered by a lack of variability in the duration of exposure. The majority of cases spent at least three hours in the office. Only four employees or visitors reported spending less then one hour in the office; two met the case definition. Thus spending as little as a half hour may have been sufficient to induce illness.

This is the first reported Pontiac fever outbreak in which antigen has been detected in the urine of cases. The sensitivity of a single urine test was low (13 per cent), but this test may be useful in the future in the early detection of cases.

The fact that only monoclonal antibody 2 was reactive in the ELISA positive urine specimens is not surprising. The antigen detected by this antibody, when present in *L. pneumophila* serogroup 1 strains, is the dominant antigen, and it consistently gives stronger readings than do the other antigens. The observed reactivity of immobilized antigens from urine with monoclonal antibody 2 is consistent with the 1,2,4,5 monoclonal reactivity pattern of the outbreak-associated isolate. Previous experience indicates that in acute-stage legionellosis, detectable antigens are present in considerably higher concentration in urine than in serum. Thus, it is not surprising that serum specimens from cases with ELISA-positive urines were negative.

As in previous outbreaks, seroconversion of epidemiologically defined cases was incomplete (21, 56, 64 per cent, and 86).[3,4,6,7] In this outbreak, 42 per cent of cases showed a four-fold or greater rise to 1:128 despite a sufficient mean interval between collection of sera of nearly six weeks.

We attempted to eliminate the organism from the cooling tower by hyperchlorination. Within 24 hours of this process, the number of organisms cultured from the cooling tower water dropped four orders of magnitude. The effect of quaternary ammonium compounds on legionellae is uncertain, but the number of organisms cultured was initially higher following this treatment. A day later, with no additional treatment, we were unable to culture *Legionella* from the tower.

Maintenance hyperchlorination was effective in that we were unable to culture organisms throughout the summer. However, the corrosive effect of chlorine on the tower may be costly. Another preventive measure would be to relocate the air intake source for the office at a point much farther than four meters from the cooling tower exhaust.

From April 26 (two days before the first exposure day) to April 30, outside air temperatures were above normal, with highs from 20 to 26 degrees Celsius. It is possible that the increased temperature of cooling tower water encouraged the growth of legionellae. This may explain the occurrence of Pontiac fever at this time. Two previous reports of Pontiac fever occurred in the spring.[5,7] These involved indoor whirlpool exposure unrelated to the outside air temperature. The remaining three reported outbreaks occurred in the summer.[3,4,6]

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UCSF Offers New Educational Program in Health Science and Human Survival

The School of Medicine of the University of California, San Francisco has just opened to the public a new educational program that explores large scale threats to human populations. The new program, called Health Science and Human Survival, brings distinguished scholars to San Francisco to speak on such man-made threats as nuclear war and the destruction of the environment. The program is the first of its kind in an American medical school.

The fundamental premise of the program is that the University and the public must work more closely together to meet the overwhelming threats to mankind posed by nuclear arms, widespread destruction of the environment, poverty, and conventional war. While such issues are to some extent political, technical, and economic problems, they are also medical, public health, and psychosocial problems, and the health science community can contribute much to public awareness and creative efforts toward their solution.

The first annual Distinguished Lecture in Health Science and Human Survival will take place on the San Francisco campus May 19, 1987. Dr. Alexander Leaf, Professor and Chair of the Department of Preventive Medicine and Clinical Epidemiology at Harvard Medical School, will talk about new findings on the chances of surviving nuclear war. Major recent findings on the biomedical effects of radiation, and on ecological effects of nuclear explosions, have not yet received public attention. For example, there is growing evidence that moderate doses of radiation often suppress immune system functioning, producing effects similar to those of AIDS (acquired immune deficiency syndrome).

Ongoing projects include formal courses and course modules in the School of Medicine, and monthly seminars, led by distinguished visitors, which bring together faculty from the UCSF campus and leaders from the surrounding community. The program also serves as a resource center for faculty and student research, and will disseminate findings and lectures to the community and other health science campuses.

The UCSF program is interested in promoting national and international exchanges on health science issues in human survival, and would welcome communications in regard to research, teaching and curricula, public action, and financial support related to this topic.

The Program in Health Science and Human Survival is administered in the Department of Epidemiology and International Health of the School of Medicine. Partial funding is provided by the University of California Institute on Global Conflict and Cooperation. Please send communications to: Christie W. Kiefer, Director, Program in Health Science and Human Survival, University of California, CSBS 237, 1350 7th Avenue, San Francisco, CA 94143. Tel: (415) 476-7543.